Psychological Medicine, 2004, **34**, 1483–1493. © 2004 Cambridge University Press
DOI: 10.1017/S0033291704002843 Printed in the United Kingdom

Juvenile- *versus* adult-onset depression: multiple differences imply different pathways

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ABSTRACT

Background. Several sources of heterogeneity in major depression have been identified. These include age of onset, presence of co-morbid disorders, and history of childhood sexual abuse. This study examined these factors in the context of the contrast between onset of depression in young women before and after age 16.

Method. Sampling was carried out in two phases. In the first, questionnaires were sent to women aged 25–36 in five primary care practices. Second-phase subjects for interview ($n = 197$) were drawn from three strata defined on the basis of childhood adversities. Interviews conducted and rated independently assessed (1) recalled childhood experiences, psychopathology and parental psychiatric disorder, and (2) adult personality functioning and adult lifetime psychopathology. Frequencies of predictor and response variables, effect estimates and their confidence intervals were weighted back to the general population questionnaire sample.

Results. Compared with adult-onset depression, juvenile-onset adult depression was associated with co-morbid childhood psychopathology and peer problems, poor parental care, and childhood sexual abuse involving actual or attempted intercourse; in adult life there were higher levels of co-morbid psychiatric disorders, and personality dysfunction. The adult-onset depression group was characterized by a history of contact childhood sexual abuse without actual or attempted intercourse, and to a lesser extent, poor parental care.

Conclusions. The juvenile- *versus* adult-onset distinction appears to be important to heterogeneity in adult depression, implicating different individual and environmental factors during childhood, and different mechanisms in adult life.

INTRODUCTION

Although effective treatments for major depression in adults over the short to medium term have been identified, it has become increasingly apparent that it is often a chronic or relapsing condition (Judd, 1997). The search for better treatments requires an adequate characterization of the causal processes in onset and recurrence. Many have been identified for depression in general; however, recent evidence suggests that different processes may operate depending on the specific features of the depression. Possible sources of heterogeneity include number of previous episodes, genetic risk, history of childhood adversities, age of onset, and patterns of co-morbidity.

Much of the work on heterogeneity has focused on variations in the role of social stressors. A reduced contribution of life events to risk for depression among those with a history of previous depression has been widely replicated (Perris, 1984; Ghazuiddin *et al.* 1990), and the association between life events and depression

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has been reported to progressively weaken with the increasing episodes (Kendler *et al*. 2000). The increasing autonomy of depressive episodes from social stressors with number of episodes has been likened to 'kindling' in epilepsy, and neurobiological and cognitive models have been proposed (Teasdale, 1988; Post, 1992; Segal *et al*. 1999; Gemar *et al*. 2001). Genetic factors may also contribute to differences in reactivity to social stressors (Kendler *et al*. 2001).

A history of childhood trauma may be a further source of heterogeneity. There is now a substantial body of work indicating that childhood sexual abuse (CSA) is an independent risk factor for major depression in women, and that it probably has a causal role (Fergusson & Mullen, 1999; Kendler *et al*. 2002; Nelson *et al*. 2002). It may be that sexual abuse is one of a number of risk factors with additive effects. An alternative possibility is that there are distinctive developmental pathways associated with CSA, in which the processes are different from those for depression associated with other factors (Kendler *et al*. 2002). We have reported previously that, in women, the association between quality of romantic relationships and depression differs depending on their childhood experiences (Hill *et al*. 2001). A history of poor parental care (PPC), characterized by neglect or institutional care, was associated with a strong link between quality of relationships and depression, mainly because there was a very high rate of depression among women who had experienced PPC and were in poor adult relationships. By contrast CSA was associated with a risk for depression that was unaltered by quality of adult relationships. This suggests different pathways in which adult interpersonal processes, and particularly attachment and cognitive processes, may play different roles. We hypothesized that women who experience CSA might use an active coping strategy to deal with the traumatic memories and their associated affects that is effective in childhood, but ineffective in the context of the demands of adult intimacy and sexuality. The precipitant for depression will be having a sexual relationship rather than its quality. Thus CSA will be associated with onset of depression in young adult life. This is in contrast to children exposed to PPC and associated family adversities, which may lead to low self-esteem, insecure attachments and helplessness, so that their vulnerability becomes evident during childhood as behavioural and emotional difficulties.

The prediction that CSA will be associated with adult-onset depression, and family adversities such as PPC with child- or adolescent-onset depression is supported by findings from the Dunedin Multidisciplinary Health and Development Study (Jaffee *et al*. 2002). On the basis of six waves of data collection up to age 26 in a representative birth cohort, 'juvenile-onset', before age 16, was found to differ from later 'adult-onset' depression in several key respects. Juvenile-onset depression was associated with an increased family history of antisocial behaviours, and with indices of family instability such as loss of a parent through death, separation or divorce, but not with CSA. The juvenile-onset group had also shown more evidence of other mental health problems in childhood and adolescence, such as conduct problems and hyperactivity, and were more likely to have been assessed as having poorer motor skills and greater temperamental inhibition. By contrast, those with adult-onset depression, compared with individuals with no history of depression, had an increased rate of CSA and residence changes, but not any of the other indices of family instability.

However, there are some indications from previous studies that the risk for psychopathology associated with CSA may vary depending on the type or extent of the abuse. Higher risk associated with actual or attempted intercourse has been reported by Mullen *et al*. (1993), Fergusson *et al*. (1996), Bulik *et al*. (2001) and Nelson *et al*. (2002). We considered that these findings might have implications for the model outlined earlier. The prediction that CSA will be associated with adult-onset depression is based on the assumption that the child has been able to use an affect regulatory coping mechanism that is, at least in childhood, effective. It could be, however, that CSA involving intercourse is so traumatic that either such a coping strategy cannot be employed, or it is overwhelmed. In which case contact CSA without intercourse should be associated with adult-onset depression, and intercourse with juvenile-onset depression.

The contrast between juvenile-onset and adult-onset depression is unlikely to be confined

to differences in childhood experiences. The Dunedin study findings suggest that adult depression with juvenile onset occurs in the context of multiple individual vulnerabilities in childhood such as poor peer relationships and inhibited temperament, and may therefore also be accompanied in adult life by a wider set of mental health problems, and greater personality dysfunction, than adult-onset depression. The idea has a long history that there may be two forms of adult depression, one 'pure' type with a family history of depression, and a 'spectrum' disorder with a family history of a broader set of psychiatric disorders such as alcoholism and antisocial personality disorder (Tsuang & Winokur, 1992; Winokur & Coryell, 1992). Associations between depression, antisocial personality disorder, and alcohol and drug abuse are also well established, and may in part reflect common genetic vulnerabilities (Fu *et al*. 2002). As far as we are aware, possible links between depression spectrum and age of onset of depression have not been previously explored.

In this paper we compare the childhood antecedents of juvenile- and adult-onset major depression, contrasting those with recalled onset before and after their 16th birthdays in a general population study of women, focusing particularly on differences in the roles of inadequate parental care, and of different types of CSA. We also examine whether there are markers for different pathways to juvenile- and adult-onset depression, and whether there is evidence that juvenile-onset adult depression, contrasted with adult-onset, is part of a wider set of episodic disorders and personality difficulties.

METHOD

Sample

Subjects were identified from among women aged 25–36 who were living on the Wirral, a borough near Liverpool in North-West England. Sampling was carried out in two phases.

In the first phase questionnaires were mailed sequentially in batches of 200–300 to all eligible women on the lists of five primary care practices. These contained an explanatory letter from the woman's primary care practitioner and questions covering adult mental health problems and childhood experiences. The 1181 returned questionnaires represented a 60·7% response rate to the first phase. A comparison of the primary care records of responders and non-responders gave no evidence of response bias with respect to mental health problems (Hill *et al*. 2000*a*). Their socioeconomic distribution was comparable to the UK as a whole (HMSO, 1993).

At the second phase, subjects were drawn for in-depth interview from the 1181. To maximize the power of the study to discriminate between the effects of childhood adversities that are often confounded in the general population, stratified random sampling was used. For the first practices, strata were defined for reported contact sexual abuse before the age of 16 (Mullen *et al*. 1993), low care (score of less than 19) on the Parental Bonding Instrument (Parker *et al*. 1979) without reported CSA, and the remainder. As a result of the low specificity of the original maternal low-care stratification criterion for the serious neglect with which we were concerned, the second stratum was redefined for sampling from the later practices to require both low maternal and low paternal care.

Once assigned, 79% agreed to take part and provided 198 completed in-depth interviews. Estimates of general population statistics were obtained using stratification weights. Weights were calculated in two steps, each being based on logistic regression models. The first was concerned with the sampling design and included stratum identifiers (which distinguished original and revised low-care strata criteria) and practice. The second was concerned with non-response after assignment, and although stratum, practice and interviewer were all included as predictors of response, only the interviewer effects were significant. The inverse of the product of the predicted probabilities from these two models was used as the combined sampling and response weight. Re-weighting the interviewed sample of 198 gave estimates for the population of women from which they were drawn as having at the time of questionnaire return a mean age of 30·8 years (S.D. = 2·9), 62% with a partner, 69% working, with a mean of 1·3 (S.D. = 1·2) children, and having come from families with 2·4 (S.D. = 1·9) siblings. The sample size reported here is 197, because one subject did not want to talk about some aspects of childhood psychopathology.

https://doi.org/10.1017/S0033291704002843 Published online by Cambridge University Press

Procedure

Women selected for phase-II interview were contacted by telephone or letter and asked to participate in the interview phase. All of the interviewers were female. The childhood and adult interviews were carried out and rated independently by two interviewers. All of the interviews were audiotaped, and ratings made from the recordings. The study was approved by the Wirral Health Authority Ethical Committee.

Measures

Recalled childhood experiences were assessed using the Childhood Experience of Care and Abuse (CECA; Bifulco *et al.* 1997). This extended investigator-based interview enquires in detail about a range of childhood experiences, including neglect, physical abuse, institutional care, sexual abuse, parental discord and partner violence. Positive ratings require specific examples of behaviors rather than mere yes/no responses to generic closed questions. Adversities are rated 'marked', 'moderate' or 'mild' and only ratings of marked or moderate are taken as denoting presence of abuse or neglect. In brief, neglect is rated on the basis of lack of attention to the child's material and emotional well-being, such as failure to provide regular meals and lack of interest in the child's life, physical abuse where the child has been hit repeatedly with a fist or an implement, and sexual abuse where there has been touching of genitals or breasts, or attempted or actual intercourse before the age of 16. Ratings of inter-parental violence require examples of at least one parent hitting the other. Parental psychiatric disorder was rated as present if the subject reported symptoms that had required treatment, or had been prolonged or associated with social impairment. No attempt was made to ascertain whether the parental disorder had met diagnostic criteria. The interviewers were trained by the principal developer of the measure (Dr Antonia Bifulco), and difficult ratings were reviewed with her regularly.

Recalled emotional and behavioural problems before the age of 16 were assessed using the Retrospective Recall of Childhood Psychopathology (RECAP; Holmshaw & Simonoff, 1996). This investigator-based interview is a modification of the Child and Adolescent Psychiatric Assessment (CAPA; Angold *et al.* 1995) and enquires about recalled psychiatric symptomatology in childhood. The interview is arranged in sections covering the major child and adolescent disorders with screening questions followed by more detailed investigation. The sections on anxiety, depression, conduct disorder and hyperactivity were used in this study. The measure was validated by comparing recalled symptoms with information abstracted from childhood psychiatric records (Holmshaw & Simonoff, 1996). Kappa coefficients based on agreement for a positive response to the screening question plus one other symptom were above 0·6 in every symptom area except hyperactivity (kappa = 0·5). In the study reported here juvenile disorder was rated where there was a positive screen plus one other symptom. Raters were trained by the authors of the RECAP and ratings were reviewed regularly.

Depression in adult life was assessed with the Schedule for Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978). The questions were adapted so that DSM-IV (APA, 1994) ratings of major depression since age 16 could be made (Hill *et al.* 2001). The onset and duration of all episodes was recorded so that they could be assigned to age periods. The interviewers were trained by J.H. who participated in the development and evaluation of the investigator-based version of the measure that was used in this study (Harrington *et al.* 1988). Ratings were reviewed regularly with J.H. All of the interviewers and raters, except RD, were not clinicians. Reliability of the ratings by the non-clinicians was checked against that of the psychiatrist (RD), from the audiotapes of 20 subjects. There was one disagreement in rating DSM major depression in the five-year periods 21–26 and 26–30 (kappa = 0·91).

Personality dysfunction was assessed using the Adult Personality Functioning Assessment (APFA; Hill *et al.* 1989). This interview enquires about functioning over time periods of 5 or 10 years in six domains of work, love relationships, friendships, non-specific social interactions, negotiations and coping. Ratings are made in each domain and over each time period on a scale from 0–5 where '0' reflects a high level of adaptation and '5' very poor functioning. This is an investigator-based measure, in which the interviewer uses flexible questioning

to obtain adequate information, and makes ratings on the basis of detailed rating rules, a dictionary of examples, and training. The APFA has good inter-rater reliability and subject-informant agreement (Hill *et al.* 1989, 1995), and pervasive dysfunction in the APFA is associated with personality disorder (Hill *et al.* 2000*b*). In this paper we have made use of ratings over the 10-year period from 21 to 30.

Variables and analyses

As the numbers who experienced institutional care were low, the neglect and in-care variables were combined to form an index of 'poor parental care' (Hill *et al.* 2001). Major depression in adult life with first episode before age 16 (juvenile-onset adult depression) was contrasted with adult depression with later onset (adult-onset major depression) in logistic regression. Each of these groups was also contrasted with adults who reported no depression since age 16. To obtain correct effect estimates and their confidence intervals required proper account to be taken of the two-phase study design (Pickles *et al.* 1995). We used inverse probability weighting to do this, as described by Dunn and co-workers (Dunn *et al.* 1999), all analyses being performed in Stata (StataCorp, 1999). Reported p values are calculated from t and F tests using the survey-based parameter co-variance matrix.

RESULTS

Seventy of the 197 interviewed women had experienced moderate or marked CSA (giving a weighted general population prevalence of 18·3%), and of these 40 had experienced contact abuse without intercourse, and 30 actual or attempted intercourse. Twenty had been abused by a parental figure. Fifty subjects described moderate or marked neglect (prevalence 14·3%), 16 reception into institutional care during childhood (prevalence 6·1%) and 68 reported physical abuse (prevalence 23·8%). Fifty-three women reported depression in one or both parents (prevalence 21·9%) and 38 reported alcoholism (prevalence 12·1%). Seventy-three women reported DSM major depression since age 16 (prevalence 22%), and of these, 29 reported juvenile onset (prevalence 8%). A further 15 women reported depression before age 16, but not subsequently (5%).

There were no differences between juvenile- and adult-onset groups in total number of episodes of depression ($p=0·75$), nor total duration of depression ($p=0·34$), but there was a trend for the juvenile-onset group to be more likely to be currently depressed ($p=0·084$). As only five women were currently depressed it was not feasible to account for current depression in the analyses; however, they were all repeated omitting these subjects. This made no difference to the results except in relation to personality functioning. Women with adult depression who reported juvenile onset differed from those with adult-onset depression in that they were more likely to have attempted suicide [odds ratio (OR) 3·0, 95% confidence interval (CI) 1·0–9·0, $p=0·06$]; to have deliberately harmed themselves without clear suicide intent (OR 25·9, 95% CI 2·8–237, $p=0·004$); to have an adult history of DSM alcohol abuse (OR 5·0, 95% CI 1·0–26·7, $p=0·06$) and to have been first pregnant as a teenager (OR 5·7, 95% CI 1·7–19·2, $p<0·005$).

Both the juvenile- and adult-onset depression groups had increased levels of dysfunction in domains of the APFA, and raised overall dysfunction scores (Table 1). However, the increased dysfunction was more severe and pervasive in the juvenile-onset group and compared with adult-onset depression was significantly higher in four of the six domains and in the overall score. In analyses omitting the five subjects with current depression the differences between juvenile- and adult-onset depression were reduced to trends in three of the four domains, and for overall dysfunction scores.

In childhood those in the juvenile-onset group were significantly more likely to have had symptoms of attention deficit and hyperactivity than those with adult-onset depression, and to have had poorer peer relationships in primary school (Table 2).

They were substantially more likely to have experienced PPC, physical abuse, and inter-parental violence. They were also much more likely than those with adult-onset depression to have been victims of CSA involving actual or attempted sexual intercourse, but not of other kinds of contact CSA. After controlling for other childhood psychopathology, the effects of PPC, physical abuse, intercourse CSA and inter-parental violence remained. When examined jointly, there were strong independent effects of

https://doi.org/10.1017/S0033291704002843 Published online by Cambridge University Press

Table 1. *Dysfunction in each of the Adult Personality Functioning Assessment domains and overall functioning, comparing adult-onset and juvenile-onset adult lifetime DSM major depression*

Domain	Adult depression (1) None: weighted mean (S.D.)	(2) Adult-onset: weighted mean (S.D.)	(3) Juvenile-onset: weighted mean (S.D.)	(1) *v.* (2)	(1) *v.* (3)	(2) *v.* (3)
Work ($n=193$)	2·18 (0·61)	2·31 (0·55)	3·25 (1·66)	0·2	0·009	0·05
Love ($n=195$)	2·97 (1·01)	3·80 (1·27)	4·07 (1·36)	0·008	0·002	0·5
Friends ($n=193$)	2·42 (0·95)	2·65 (0·95)	3·21 (1·15)	0·2	0·001	0·06
Non-specific ($n=192$)	2·47 (0·74)	2·59 (0·87)	3·39 (1·20)	0·7	<0·001	0·006
Negotiations ($n=191$)	2·27 (0·72)	2·44 (0·71)	3·04 (1·11)	0·2	0·004	0·04
Coping ($n=191$)	2·26 (0·57)	2·63 (0·91)	2·93 (1·29)	0·04	0·01	0·5
Total ($n=186$)	14·57 (3·00)	16·74 (3·31)	20·22 (6·16)	0·002	0·001	0·05

Higher scores in each domain reflect increased dysfunction. Domain scores were compared as ordinal counts with p values from ordinal logistic Wald tests. Total scores were compared using t tests.

Table 2. *Childhood risk factors comparing juvenile-onset and adult-onset adult lifetime DSM major depression* ($n=73$)

	Simple weighted odds ratio (95% CI)
Conduct	3·6 (0·8–16·3)
Hyperactivity	9·6 (2·6–35·5)***
Anxiety	2·9 (0·9–9·0)
No primary school friends	5·7 (1·6–19·9)**
Not popular at primary school	4·0 (1·3–12·4)*
Poor parental care	5·8 (1·8–19·1)**
Child sexual abuse	
Contact only	1·4 (0·4–4·9)
Intercourse	10·9 (2·5–46·9)***
Physical abuse	4·4 (1·4–13·8)**
Inter-parental violence	5·4 (1·7–16·9)**
Parental alcoholism	1·9 (0·5–7·5)
Parental depression	2·3 (0·8–7·0)

Odds ratios of greater than 1 indicate increased odds of the childhood risk factor associated with juvenile-onset depression compared with adult-onset depression.
* $p<0·05$, ** $p<0·01$, *** $p<0·001$.

Table 3. *Childhood risk factors comparing juvenile-onset adult lifetime DSM major depression and no major depression in adult life* ($n=153$)

	Simple weighted odds ratio (95% CI)
Conduct	3·43 (1·2–10·1)*
Hyperactivity	16·36 (5·6–47·8)***
Anxiety	5·46 (2·0–15·2)***
No primary school friends	12·3 (4·3–35·3)***
Not popular at primary school	5·2 (1·9–14·5)**
Poor parental care	23·29 (8·0–67·9)***
Child sexual abuse	
Contact only	12·88 (4·3–38·9)***
Intercourse	10·56 (3·06–36·5)***
Physical abuse	7·21 (2·7–19·4)***
Inter-parental violence	4·26 (1·6–11·3)**
Parental alcoholism	4·18 (1·6–11·3)**
Parental depression	7·33 (1·3–14·5)***

* $p<0·05$, ** $p<0·01$, *** $p<0·001$.

PPC (OR 7·7, 95% CI 1·4–41·2, $p=0·02$) and intercourse CSA (OR 9·1, 95% CI 1·5–54·4, $p=0·02$).

Table 3 compares women with juvenile-onset adult depression and those with no major depression in adult life. This shows the breadth of the co-morbid childhood disorders and family adversities in the juvenile-onset group. Analysis of the joint effects of childhood exposures that remained significant after controlling for childhood psychopathology suggested a major contribution from PPC (OR 9·37, 95% CI 2·6–33·9, $p=0·001$), and a significant association with parental depression (OR 3·86, 95% CI 1·3–11·5, $p=0·02$). The possibility of additional independent effects of CSA could not be convincingly excluded (CSA intercourse OR 3·86, CI 0·78–19·0, $p=0·1$; CSA contact OR 2·72, CI 0·5–14·7, $p=0·2$).

The characteristics of women with adult-onset depression are compared with those with

https://doi.org/10.1017/S0033291704002843 Published online by Cambridge University Press

Table 4. *Childhood risk factors comparing adult-onset adult lifetime DSM major depression and no major depression in adult life* ($n = 168$)

	Simple weighted odds ratio (95% CI)
Conduct	0·9 (0·3–3·5)
Hyperactivity	1·6 (0·5–5·6)
Anxiety	1·9 (0·7–5·5)
No primary school friends	2·2 (0·7–6·8)
Not popular at primary school	1·3 (0·5–3·5)
Poor parental care	4·0 (1·5–10·6)**
Child sexual abuse	
Contact only	7·7 (3·0–20·0)***
Intercourse	1·2 (0·3–4·2)
Physical abuse	1·6 (0·7–4·0)
Inter-parental violence	0·8 (0·3–2·1)
Parental alcoholism	2·2 (0·6–7·4)
Parental depression	3·2 (1·1–9·0)*

* $p < 0·05$, ** $p < 0·01$, *** $p < 0·001$.

no major depression in adult life in Table 4. The pattern of associations is quite different from that of juvenile-onset depression, in that childhood disorders and adversities, other than PPC and contact sexual abuse without actual or attempted intercourse, are conspicuously smaller and non-significant.

The childhood exposures that remained significant after controlling for childhood psychopathology suggested strong and significant independent effects for contact CSA without intercourse (OR 8·4, 95% CI 3·0–23·9, $p < 0·001$), and increasingly modest effects for poor care (OR 3·8, 95% CI 1·2–11·9, $p = 0·02$) and for parental depression (OR 2·53, 95% CI 1·11–5·77, $p = 0·03$). These three remained significant when considered jointly (CSA contact only: OR 7·7, 95% CI 2·3–26·1, $p = 0·001$; PPC: OR 3·7, 95% CI 1·2–12·0, $p = 0·03$; parental depression: OR 3·1, 95% CI 1·0–9·3, $p = 0·04$). By contrast there were no indications of a contribution from CSA involving intercourse.

DISCUSSION

These findings provide further support for the validity of the distinction between juvenile- and adult-onset depression, and they are consistent with our previous speculation contrasting the impact of CSA and parental neglect and institutional care. Compared with subjects with DSM major depression whose first episode occurred after age 16, those with adult depression preceded by depression in childhood or early adolescence (juvenile-onset) differed in their rates of co-morbid childhood disorders, childhood adversities, adult psychiatric disorders and personality dysfunction. Comparisons of each of the juvenile- and adult-onset groups with women reporting no episodes of major depression in adult life confirmed these differences. However, there did appear to be a role for childhood adversities in adult-onset depression, specifically for contact CSA not involving actual or attempted intercourse.

The main methodological limitation of the study was the reliance on retrospective methods for ascertaining childhood experiences and psychopathology. The use of retrospective measures of childhood adversity has been criticized on several grounds (Maughan & Rutter, 1997), notably that adults without psychiatric disorders are more likely to underestimate childhood adversities. Nevertheless studies of adults with known abuse histories (Williams, 1994), of siblings' reports (Bifulco *et al.* 1997; Duggan *et al.* 1998) and of genetic influences on reports of parenting (Kendler, 1996) suggest that reports of major childhood adversities are reasonably robust. Retrospective reports may be more satisfactory where serious adversities (contrasted with more subtle variations in parenting), are ascertained from detailed interviews that require subjects to provide examples of behaviour (Hardt & Rutter, 2004). The quality of recall of adult lifetime psychiatric disorder is variable (Andrews *et al.* 1999), although test–retest reliability for depression over similar time periods to those in this study has been moderate to good (Prusoff *et al.* 1988; Kendler *et al.* 1993). Holmshaw & Simonoff (1996) reported high positive predictive values for anxiety, depression and conduct disorder with the RECAP measure used in this study. However, that was based on a clinic population and generalization to the general population cannot be assumed. The ascertainment of parental psychiatric disorder was also limited in that no other family members were interviewed, and the diagnostic status of the reported disorders was not established.

Nevertheless the consistency of the findings from this and the prospective Dunedin study (Jaffee *et al.* 2002) are striking and this provides support for the validity of the retrospective methods used here. In both studies co-morbid

childhood disorders were associated with juvenile-onset depression. We found that PPC, mainly comprising parental neglect, was the strongest discriminator between juvenile-onset and adult-onset depression, and in the Dunedin study several measures of family instability were associated with juvenile-onset depression. These included number of parent figure changes and loss of a parent through death, divorce or separation, which are likely to have been markers for the experiences that were assessed retrospectively in this study. In particular, parental neglect has been found to mediate, and to a certain extent explain, associations between parental loss and depression (Harris *et al.* 1986). Further support for the validity of the measures in this study comes from comparison with prevalence rates reported previously. The estimates of the prevalence of histories of CSA obtained from the questionnaires (17·5%), and from the weighted interview data (18·6%) were close to the weighted average estimate of 19·2% reported by Fergusson and Mullen (1999) on the basis of studies published up to 1997. The estimated population prevalence of DSM-IV major depression since age 16 in our study (22·0%) was consistent with the range reported from USA epidemiological studies. In the Epidemiological Catchment Area Study (ECA) the lifetime prevalence of DSM-III major depression in women aged 18–29 was 10·6%, and in those aged 30–44 it was 15·3% (Weissman *et al.* 1991). The estimate for DSM-III-R major depression in women aged 15–54 in the National Co-morbidity Study was 21·3% (Kessler *et al.* 1994). Finally, the investigator-based interviews used in this study are designed to provide ratings on the basis of descriptions of behaviours, and not evaluations or summary statements by the subject. The comparisons of the prevalence rates of child maltreatment and adult depression between this and other studies are also relevant to consideration of whether the sample was representative of the general population. The return rate of the questionnaires was modest, and this may have introduced bias, although the comparable prevalence rates suggest that simple biases, arising for example from maltreated women being more or less likely to return questionnaires, were probably not occurring. However, we cannot rule out other forms of bias, such as that women with a history of depression and child maltreatment were more likely to return their questionnaires than those with depression in the absence of child maltreatment.

The findings of associations between childhood maltreatment and adult depression are consistent with numerous recent reports (Fergusson & Mullen, 1999). Generally it has not been possible to determine from these the extent to which the associations provide evidence for a causal role for childhood adversities or reflect correlated genetic influences. Nelson *et al.* (2002) reported findings from a telephone-interview-based study in Australia of 1991 twin pairs, with measures of CSA, physical abuse, neglect, parental conflict, and step-parent presence. After controlling for other childhood adversities twins who reported CSA had higher rates of a range of adult psychiatric disorders, including DSM major depression, than their CSA-negative co-twins of the same sex. Kendler *et al.* (2002) used data from 1942 adult female twin pairs to construct a developmental model for adult depression. Structural equation modelling including estimates of genetic and environmental risks, indicated an independent role for CSA in increasing risk of DSM major depression.

A further way of examining whether CSA may have a causal role in depression is to test specific hypotheses that predict differential effects, in this case contrasting contact sexual abuse with and without intercourse in relation to age of onset of depression. The idea that the effects of CSA may not be apparent in childhood, but are seen in adult life when they are activated by the demands of adult social roles, goes back to Freud (1961–63). For Freud the key processes were repression and dissociation, which find their counterparts in concepts of biases or distortions within modern cognitive science (Power & Brewin, 1991). Biases in autobiographical memory may be relevant to links between CSA and depression. Williams (1996) proposed that the tendency to favour general (categoric) over specific autobiographical memories, found in individuals with histories of depression and attempted suicide, may have arisen in childhood as an affect regulatory strategy in the face of trauma. Were this the case, then affective symptoms would not be expected over the developmental phase in which

the coping strategy was implemented, but might be evident later when the strategy was no longer effective. The proposition that sexually abused children are *unlikely* to have affective symptoms would come as a surprise to many clinicians, and would seem to be at odds with the considerable evidence that CSA is associated with post-traumatic stress symptomatology (see, for example, Wolfe *et al*. 1994; Wekerle *et al*. 2001). There are, however, indications that sexually abused children may have raised *or* reduced levels of symptoms. For example, Elliott & Briere (1994), in a study of children referred to child protection agencies, found that sexually abused children who disclosed abuse had higher levels of post-traumatic stress symptomatology than non-abused children, but children who had been abused (according to objective evidence) but had not disclosed had lower levels than non-abused children. The authors suggest that non-disclosure reflected the children's use of a self-protective form of denial that was effective in reducing painful affects. Such a polarization into high and low symptoms groups is consistent with our interpretation of the findings in this study, based on the prediction that a coping strategy can be put in place where the abuse is not overwhelming. This distinction appeared to be captured by whether or not the abuse involved actual or attempted sexual intercourse. There were indications that this was also the case in findings reported by Fergusson *et al*. (1996). All forms of contact CSA reported at age 18 were associated with major depression between ages 16 and 18, with a higher risk associated with intercourse than other forms of contact. However, only CSA involving intercourse was associated with major depression between ages 14 and 16. Equally, it is likely that other factors requiring prospective study are also involved.

The difference between juvenile- and adult-onset depression is not only one of timing of onset. In the group with juvenile onset, depression in adult life was associated with self-harm and alcohol problems, and with dysfunction across six domains of interpersonal and social role functioning. The differences between the juvenile- and adult-onset groups shown in Table 1 were attenuated when five subjects with current depression were omitted from the analyses. This may reflect an effect of current depression on reporting of personality dysfunction, and so the conclusions discussed here must be seen as tentative. However, indications of increased levels of adult personality dysfunction in the juvenile-onset adult depression, taken together with the breadth of social dysfunction in childhood reported here, and in the study by Jaffee *et al*. (2002), imply that the group is characterized by quite pervasive dysfunction that persists from childhood to adult life. Person–environment interactions are implied by our finding that teenage pregnancy is associated with juvenile- but not adult-onset depression. Pathways from childhood institutional care, neglect, and childhood conduct disorder, via early unplanned pregnancy, in turn increasing the risk for unsupportive cohabitations and for subsequent adult psychopathology, are well described (Harris *et al*. 1987; Quinton *et al*. 1993). Whether such pathways apply only to juvenile-onset depression requires further investigation.

It will be important in future studies to avoid the assumption that these and previous data imply two different syndromes. Rather they point to a set of contrasting developmental processes in the genesis of depression in adult life that need further investigation. It is not yet clear whether the juvenile- *versus* adult-onset distinction, taking a threshold of 16, is a subset of a broader set of differences in depression depending on age of onset. It is likely that juvenile-onset depression as defined here is itself heterogeneous, given the substantial impact of puberty on the incidence of depression (Angold *et al*. 1998; Weissman, 2002). Jaffee *et al*. (2002) combined males and females in their analyses and we studied only women, so that gender differences or similarities in the juvenile- *versus* adult-onset distinction remain to be explored. Crucially, there may be further distinctions to be made in adult life. Our hypothesis is that the risk for adult-onset depression associated with contact CSA arises because individuals are vulnerable when they start to have adult romantic relationships involving sexual intimacy, and so we do not expect that CSA will be associated with onset in middle age. There may be other vulnerabilities for depression arising from genetic or early childhood experiences that come 'on line' later, or ones that have nothing to do with these factors.

ACKNOWLEDGEMENT

The work was supported by an MRC grant to Professor Jonathan Hill (grant no. G9304757).

DECLARATION OF INTEREST

None.

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